Verapamil-sensitive left fascicular ventricular tachycardia (FVT) has been shown to be a reentrant mechanism using the Purkinje network as a part of its reentrant circuit. Although the papillary muscles (PMs) are implicated in arrhythmogenic structure, reentrant FVT originating from the PMs has not been well defined.

Methods and Results—We studied 13 patients in whom FVT was successfully eliminated by ablation at the posterior PMs (n=8; PPM-FVT) and anterior PMs (n=5; APM-FVT). Although intravenous administration of verapamil (5 mg) terminated ventricular tachycardia (VT) in 6 patients, VT was only slowed in the remaining 7 patients. PPM-FVT exhibited right bundle branch block and superior right axis (extreme right axis) or horizontal axis deviation. APM-FVT exhibited right bundle branch block configuration and right axis deviation with deep S wave in leads I, V5, and V6. VT was reproducibly induced by programmed atrial or ventricular stimulation. His-ventricular interval during VT was shorter than that during sinus rhythm. Ablation at the left posterior or anterior fascicular regions often changed the QRS morphology but did not completely eliminate it. Mid-diastolic Purkinje potentials were recorded during VT around the PMs, where ablation successfully eliminated the tachycardia. All patients have been free from recurrent VT after ablation.

Conclusions—Reentrant circuit of verapamil-sensitive FVT can involve the Purkinje network lying around the PMs. PVM-FVT is a distinct entity that is characterized by distinctive electrocardiographic characteristics and less sensitivity to verapamil administration compared with common type FVT. Ablation targeting the mid-diastolic Purkinje potentials around the PMs during tachycardia can be effective in suppressing this arrhythmia.

Key Words: catheter ablation • fascicular ventricular tachycardia • papillary muscles


**WHAT IS KNOWN**

- Verapamil-sensitive left fascicular ventricular tachycardia (FVT) is a reentrant mechanism using the Purkinje network as a part of its reentrant circuit.
- According to the QRS morphology and the site of origin, verapamil-sensitive FVT has been classified into 3 patterns: left posterior type, left anterior type, and upper septal type.

**WHAT THE STUDY ADDS**

- Reentrant circuit of verapamil-sensitive FVT can involve the Purkinje network lying around the papillary muscles.
- This subtype of FVT is another identifiable entity, which is characterized by distinctive electrocardiographic characteristics and less sensitivity to verapamil administration compared with the common type FVT.
- Ablation targeting the diastolic Purkinje potentials that are recorded around the papillary muscles during tachycardia can be effective in suppressing this arrhythmia.

with repetitive premature ventricular contractions or nonsustained ventricular tachycardia (VT) in the absence of sustained VT were excluded. Structural heart diseases were ruled out by 12-lead ECG, chest radiographs, echocardiography, cardiac computed tomography when appropriate, and coronary angiography. All patients had no evidence of structural heart disease, as determined by a normal ejection fraction, no history of congestive heart failure, and no evidence of coronary artery disease. This study complied with the guidelines of the Declaration of Helsinki and was approved by the institutional review board of Tsukuba University Hospital. All patients gave written informed consent before catheter ablation.

**Twelve-Lead ECG**

Twelve-lead ECG was recorded digitally at a sweep speed of 100 mm/s for offline analysis. The analysis of 12-lead ECG focused on the after characteristics: bundle branch block morphology (right or left), axis, QRS duration, and morphology of the QRS complex. The QRS duration was measured as the interval between the earliest onset of the QRS complex in any lead and the latest offset in any lead on the 12-lead ECG.

**Electrophysiological Study**

An electrophysiological study was performed after all antiarrhythmic drugs had been discontinued. Five-French quadripolar catheters were inserted from the right femoral vein and placed at the His-bundle region and the right ventricular apex. The left ventricular (LV) endocardium was accessed by a retrograde transaortic approach. A 4-mm-tip nonirrigated catheter (Navistar; Biosense Webster, Diamond Bar, CA) or irrigation catheter (Navistar ThermoCool; Biosense Webster) was used for mapping the LV, pacing, and ablation. Bipolar signals were filtered from 30 to 500 Hz. An intracardiac echocardiography catheter (SoundStar; Biosense Webster) and the CartoSound system (Biosense Webster) were used to create a 3-dimensional (3D) reconstruction of the LV, including the PMs by tracing endocardial surface contours. The exact location of mapping/ablation catheter was confirmed by real-time intracardiac ultrasound image.

Programmed ventricular stimulation was performed from the right atrium and right ventricular apex at basic drive cycle lengths of 400 ms with up to triple extrastimuli decrementing down to 180 ms or to refactoriness, whichever occurred first. If sustained VT was not induced, the programmed stimulation was repeated under isoproterenol infusion (0.5–2 μg/min). When VT was induced, LV mapping was performed searching for diastolic and presystolic Purkinje potentials. Entrainment of the VT by pacing from the right atrium, right ventricular apex, and successful ablation site in the LV was confirmed, although dependent on the operator’s preference.

**Radiofrequency Ablation**

Radiofrequency energy was delivered with a maximum power of 50 W and a target temperature of 42°C for the irrigation catheter and 55°C for the nonirrigated catheter. The current was applied during VT to the site showing Purkinje potentials. When both diastolic and presystolic Purkinje potentials (P1 and P2) were observed, ablation was initially done at the most distal site where diastolic and presystolic Purkinje potentials were sequentially recorded to minimize the risk of inadvertent left bundle branch block. If the VT terminated within 20 s, additional current was delivered for ≤120 s. If the VT was not terminated, ablation was directed to a more proximal site with the earlier diastolic potential. After termination of VT, programmed electrical stimulation was repeated, using the same protocols as preablation induction with and without isoproterenol infusion. The procedural end point of ablation was both the lack of VT inducibility and no ventricular echo beat identical to the clinical VT morphology.

**Postablation Management**

Patients were monitored at least 24 hours before discharge. No antiarrhythmic drugs were prescribed after ablation procedure. Patients were followed through regular clinic visits the first month and every 3 months thereafter. A 24-hour Holter recording was obtained at approximately yearly intervals. Arrhythmia recurrence was assessed by patient interview, 12-lead ECG, and Holter recording.

**Statistics**

Categorical variables were expressed as numbers and percentages. Continuous data were expressed as mean±SD and assessed by the Wilcoxon rank-sum test. All tests were 2-tailed, and a probability value of <0.05 was considered statistically significant.

**Results**

**Clinical Characteristics**

Baseline characteristics of 13 patients are shown in Table 1. There were 11 men and 2 women (37±16 years of age). All had normal LV systolic function, and there was no evidence of structural abnormality. The presenting arrhythmia was sustained monomorphic VT in all patients. Eleven patients presented with palpitations, and 2 presented with presyncope. Ten patients had undergone previous ablation procedures. In the initial procedure, RF energy was delivered at the left posterior or anterior fascicular region of the LV septum. In 4 patients, the LV false tendon was detected by transthoracic echocardiography or cardiac computed tomography. VT was not responsive to adenosine bolus in all of 7 patients tested.

Although the baseline ECG exhibited sinus rhythm with QRS duration <120 ms and PQ interval <200 ms in all patients, a small q wave in the inferior limb leads was found in 5 patients. In 4 patients, this minor morphological change, that could indicate conduction abnormality in the left posterior fascicle, developed after the initial ablation. In 1 patient who had undergone previous ablation at the left anterior fascicular region, the baseline ECG exhibited left axis deviation, suggesting conduction abnormality in the left anterior fascicle.
Verapamil Sensitivity
Intravenous administration of 5-mg verapamil terminated VT in 6 patients. In the remaining 7 patients, VT was slowed by 5-mg verapamil injection but was not terminated. In 4 of these patients, VT was terminated by 10-mg verapamil injection.

Electrocardiographic Findings
Figure 1 showed the QRS morphology of PM-FVT preablation in all patients. The mean QRS duration during VT was 128±8 ms (Table 2). There was no significant difference in QRS duration between PPM-FVT and APM-FVT (126±8 versus 131±7 ms; P=0.21). PPM-FVT exhibited right bundle branch block (RBBB) configuration with superior right axis deviation (extreme right axis) deviation in 7 patients and horizontal axis deviation in 1 patient. All patients with PPM-FVT had a small r wave in lead III. In all patients with PPM-FVT except one, rS pattern with R/S ratio <1 was observed in leads I, V5, and V6. APM-FVT exhibited RBBB configuration and right axis deviation in 7 patients and horizontal axis deviation in 1 patient. In 1 patient, atrioventricular nodal reentrant tachycardia converted to VT. During the initial VT, retrograde activation of the His-bundle was recorded after the onset of QRS complex in all patients. There was no significant difference in His-ventricular interval during VT between PPM-FVT and APM-FVT (−15±7 versus −17±8 ms; P=0.66). As shown in Figure 5, diastolic and presystolic Purkinje potentials (P1 and P2) were sequentially recorded during VT. P1 activated from the proximal to distal electrode, whereas P2 activated in the reverse direction. The earliest Purkinje potential preceded QRS onset by an average of 48±18 ms. Entrainment pacing was performed in 8 patients and demonstrated concealed fusion in 2 patients. In 6 patients, selective capture of the Purkinje potential was not obtained.

Electrophysiological Findings and Ablation Outcome
Procedural outcomes were summarized in Table 3. VT was reproducibly induced by atrial pacing in 7 patients and by programmed ventricular extrastimulation in 8 patients. In 1 patient, atrioventricular nodal reentrant tachycardia converted to VT. During the initial VT, retrograde activation of the His-bundle was recorded after the onset of QRS complex in all patients. There was no significant difference in His-ventricular interval during VT between PPM-FVT and APM-FVT (−15±7 versus −17±8 ms; P=0.66). As shown in Figure 5, diastolic and presystolic Purkinje potentials (P1 and P2) were sequentially recorded during VT. P1 activated from the proximal to distal electrode, whereas P2 activated in the reverse direction. The earliest Purkinje potential preceded QRS onset by an average of 48±18 ms. Entrainment pacing was performed in 8 patients and demonstrated concealed fusion in 2 patients. In 6 patients, selective capture of the Purkinje potential was not obtained.

Successful ablation site was located around the PMs in all patients. In 11 patients, ablation of the earliest Purkinje potential during VT terminated the tachycardia and rendered VT noninducible. In the remaining 2 patients (patient numbers 2 and 10), VT was terminated by mechanical manipulation of the ablation catheter that was located at the PMs where Purkinje potentials were recorded. Thereafter, any pacing maneuvers failed to induce the VT that was reproducibly

Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Sex</th>
<th>LVEF (%)</th>
<th>No. of Previous Ablation</th>
<th>ECG in Sinus Rhythm</th>
<th>Response to Verapamil 5 mg Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>33</td>
<td>F</td>
<td>60</td>
<td>3</td>
<td>Small q wave in inferior leads</td>
<td>VTCL prolongation</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>F</td>
<td>69</td>
<td>1</td>
<td>Small q wave in inferior leads</td>
<td>VT termination</td>
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<tr>
<td>3</td>
<td>34</td>
<td>M</td>
<td>63</td>
<td>0</td>
<td>Small q wave in inferior leads</td>
<td>VTCL prolongation</td>
</tr>
<tr>
<td>4</td>
<td>39</td>
<td>M</td>
<td>73</td>
<td>3</td>
<td>Small q wave in inferior leads</td>
<td>VT termination</td>
</tr>
<tr>
<td>5</td>
<td>64</td>
<td>M</td>
<td>63</td>
<td>1</td>
<td>Small q wave in inferior leads</td>
<td>VT termination</td>
</tr>
<tr>
<td>6</td>
<td>29</td>
<td>M</td>
<td>66</td>
<td>1</td>
<td>Normal</td>
<td>VTCL prolongation</td>
</tr>
<tr>
<td>7</td>
<td>40</td>
<td>M</td>
<td>65</td>
<td>1</td>
<td>Normal</td>
<td>VTCL prolongation</td>
</tr>
<tr>
<td>8</td>
<td>46</td>
<td>M</td>
<td>70</td>
<td>0</td>
<td>Normal</td>
<td>VTCL prolongation</td>
</tr>
<tr>
<td>9</td>
<td>70</td>
<td>M</td>
<td>61</td>
<td>2</td>
<td>Small q wave in inferior leads</td>
<td>VTCL prolongation</td>
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<tr>
<td>10</td>
<td>42</td>
<td>M</td>
<td>61</td>
<td>0</td>
<td>Normal</td>
<td>VTCL prolongation</td>
</tr>
<tr>
<td>11</td>
<td>30</td>
<td>M</td>
<td>69</td>
<td>1</td>
<td>Normal</td>
<td>VTCL prolongation</td>
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<tr>
<td>12</td>
<td>26</td>
<td>M</td>
<td>57</td>
<td>1</td>
<td>Normal</td>
<td>VTCL prolongation</td>
</tr>
<tr>
<td>13</td>
<td>22</td>
<td>M</td>
<td>65</td>
<td>2</td>
<td>Left axis deviation</td>
<td>VT termination</td>
</tr>
</tbody>
</table>

F indicates female; LVEF, left ventricular ejection fraction; M, male; and VTCL, ventricular tachycardia cycle length.
inducible before the mapping catheter was moved to the site. At the end of procedure, VT became noninducible in all cases.

The mean procedure time was 178±33 minutes, with a mean radiofrequency energy delivery time of 21±11 minutes. Neither atrioventricular block nor left bundle branch block was seen during or after the ablation procedure. Patients were followed for 42±31 months without antiarrhythmic drugs. All patients were free from recurrent VT during the follow-up period.

Discussion

Major Findings

Reentrant circuit of verapamil-sensitive FVT can involve the Purkinje network lying around the PMs. This is the first report to demonstrate a distinct subtype of verapamil-sensitive FVT originating from the Purkinje network around the PMs. According to the QRS morphology and the site of origin, verapamil-sensitive FVT has been classified into 3 patterns. The most common type is left posterior FVT, in which QRS morphology exhibits RBBB configuration and left axis deviation.5 Left anterior FVT exhibits RBBB configuration and right axis deviation.6 Upper septal FVT, which is the most uncommon variant of FVT, exhibits a narrow QRS configuration and normal or right axis deviation.16 This study demonstrates that PM-FVT is another identifiable verapamil-sensitive FVT (Figure 6), which is characterized as follows;

1. PPM-FVT exhibited RBBB configuration and superior right axis deviation or horizontal axis. APM-FVT exhibited RBBB configuration and right axis deviation with a deep S wave in leads I, V5, and V6.

2. Although PM-FVT is sensitive to verapamil administration, it is not as highly sensitive as the common type of left FVT.

3. Ablation at the left posterior or anterior fascicular regions often changes the QRS morphology and cycle length of PM-FVT but does not completely eliminate it.

4. Diastolic Purkinje potential during tachycardia is recorded at the PMs during VT. Ablation of the diastolic potentials is highly effective for suppressing this arrhythmia.
Although the QRS morphology of PM-FVT has many similarities to that of left posterior and anterior FVT, there are distinguishable differences. All PM-FVT exhibits RBBB configuration, but superior right axis or horizontal axis deviation in PPM-FVT and R/S ratio <1 in leads I, V5, and V6 in APM-FVT is a predictor for differentiating PM-FVT from common type FVT.

The critical limb of the reentrant circuit of left FVT incorporates the specialized Purkinje tissue, generating a diastolic potential during tachycardia that lies in a zone of slow conduction with decremental properties and verapamil sensitivity. The diastolic Purkinje potential during common type FVT is recorded at the distal third of the fascicle-Purkinje network on the LV septum. The relative activation times of mid-diastolic Purkinje potentials during FVT in previous studies range from 30 to 70 ms pre-QRS. In contrast, mid-diastolic Purkinje potentials during PM-FVT were not recorded at the left fascicular regions on the LV septum, but around the PMs. During PM-FVT, the activation propagated antegradely from the basal to apical site of the PMs, which generated mid-diastolic Purkinje potentials. Presystolic Purkinje potentials were subsequently recorded with a distal to proximal activation sequence. The sequential activation pattern of diastolic and presystolic Purkinje potentials during PM-FVT is consistent with that during common type FVT.

It is noteworthy that QRS morphology and VT cycle length of PM-FVT often changed after ablation of left posterior or anterior fascicle regions where presystolic Purkinje potentials during PM-FVT were recorded. Ablating distally the presystolic Purkinje potential during PM-FVT did not result in complete suppression of arrhythmia. This suggests that ablation of the left fascicle region may abolish both a secondary exit and part of the reentry circuit but does not eliminate the critical limb of the reentry circuit. Because the PMs are anatomically complex structures with variable distribution of Purkinje fibers, there is the potential for variable recruitment of the Purkinje network for the reentry circuit of PM-FVT. FVT may be able to persist by using interlinking Purkinje network fibers after ablation of the noncritical component of its reentrant circuit.

The reason for less sensitivity to the verapamil administration is undetermined from our data. Although the nature of the reentry circuit of FVT has not been fully elucidated, the antegrade limb of the FVT circuit that is represented as the mid-diastolic potential (P1) is thought to have decremental properties and verapamil sensitivity. A previous study suggested that the verapamil-sensitive zone seemed to be located upstream of the FVT circuit that may be more proximal to P1. We can speculate that the critical limb of the PM-FVT circuit may involve fewer proximal elements of the Purkinje network. Further studies to clarify the pharmacological effect of intravenous verapamil on PM-FVT circuit are warranted.

Relevance of Fibromuscular False Tendon to PM-FVT

Previous studies reported that the fibromuscular false tendon may be associated with FVT because ablation or surgical removal of the false tendon results in suppression of FVT in some cases. A fibromuscular false tendon is a fine muscular strand which carries Purkinje fibers and may traverse the LV cavity between the PMs and the intraventricular septum. It is possible that in some cases of PM-FVT, there is an anatomical and electrical connection between the APM and PPM, which may explain the changes in QRS morphology from superior to inferior axis deviation during ablation. In fact, Figure 7 shows an autopsy specimen of the human heart that illustrates the myocardial structure between the

<table>
<thead>
<tr>
<th>Table 2. ECG Characteristics</th>
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<tbody>
<tr>
<td>Patient No.</td>
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<tr>
<td>1</td>
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<td>2</td>
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<td>3</td>
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<td>11</td>
</tr>
<tr>
<td>12</td>
</tr>
<tr>
<td>13</td>
</tr>
<tr>
<td>Mean±SD</td>
</tr>
</tbody>
</table>

APM indicates anterior papillary muscle; PPM, posterior papillary muscle; RBBB, right bundle branch block; and VTCL, ventricular tachycardia cycle length.
APM and PPM. However, in this study, intracardiac echocardiography was not always able to give evidence supporting an association between the fibromuscular false tendon and PM-FVT. The reentrant circuit of PM-FVT comprised the Purkinje network lying on the small anatomic structures such as fibromuscular bands, trabeculae carneae, and complex arrangements of small PMs, which were not always visualized by intracardiac echocardiography.

Differentiation From Ventricular Arrhythmias Originating From the PM Myocardium
PM-FVT may be confused with idiopathic ventricular arrhythmias originating from the PM myocardium. There are several clinical and electrophysiological hallmarks to differentiate PM-FVT from idiopathic arrhythmias originating from the PM myocardium. First, the majority of patients with ventricular arrhythmias originating from the PM myocardium present with premature ventricular beats with a nonreentrant focal mechanism, not sustained VT, as their clinical arrhythmia.9,14,21,22 Second, idiopathic PM arrhythmias are refractory to verapamil. Third, electrophysiological findings, including inducibility with atrial and ventricular extrastimulation, entrainment, and diastolic Purkinje potential during tachycardia are uniformly consistent in patients with PM-FVT, which indicates that a reentrant mechanism with the circuit incorporating that Purkinje fibers is proposed as the mechanism of PM-FVT.

The differentiation of PM-FVT from arrhythmias originating from PM myocardium on the basis of ECG characteristics may be challenging. In fact, ECG characteristics of PM-FVT are similar to those of ventricular arrhythmias from PM myocardium. Moreover, varying QRS morphology during ablation is also occasionally observed in PM ventricular arrhythmias.21 However, one electrocardiographic clue to assist in differentiating these arrhythmias may be the QRS duration during ablation. Previous studies reported that PM ventricular arrhythmias were distinguished by longer QRS durations. On the contrary, the mean QRS width of PM-FVT in this study was 125±6 ms, which is much narrower than that of PM ventricular arrhythmias.

Catheter ablation of ventricular arrhythmias originating from the PM myocardium is often challenging and
is likely to have a lower success rate than ablation of LV fascicular arrhythmias.9 This is in part because of the difficulty in maintaining stable contact of the catheter tip with PMs and the deep intramyocardial origin of PM ventricular arrhythmias.21 On the other hand, the critical elements of the reentrant circuit of PM-FVT might be the Purkinje network located at a more superficial layer, although it depends on the degree of endocardial penetration of the Purkinje fibers.

**Figure 3.** Change in QRS morphology from superior to inferior axis deviation. **A,** Ventricular tachycardia (VT) at baseline (VT1) exhibited right bundle branch block (RBBB) configuration and superior right axis deviation. Radiofrequency ablation was delivered at the left ventricular (LV) posterior fascicular region where presystolic Purkinje potential was recorded. However, VT was still inducible and changed its morphology after the ablation. **B** and **C,** The final successful ablation site was on the papillary muscle where diastolic P1 potential was recorded (arrow), which preceded the QRS onset by 65 ms (patient number 6).
This is supported by the fact that premature termination and subsequent noninducibility may sometimes occur during mapping of PM-FVT. Careful catheter manipulation so as not to mechanically render the VT noninducible is important.

Table 3. Procedure Outcome

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Mode of VT Induction Preablation</th>
<th>H-V Interval in SR, ms</th>
<th>H-V Interval in VT, ms</th>
<th>Earliest PP-QRS in VT, ms</th>
<th>VT Inducibility Postablation</th>
<th>Follow-Up Duration</th>
<th>Recurrence</th>
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<tr>
<td>1</td>
<td>Ventricular extrastimulation</td>
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<td>−16</td>
<td>48</td>
<td>No</td>
<td>46</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>Atrial pacing, ventricular extrastimulation</td>
<td>30</td>
<td>−14</td>
<td>32</td>
<td>No</td>
<td>51</td>
<td>No</td>
</tr>
<tr>
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<td>Ventricular extrastimulation</td>
<td>34</td>
<td>−18</td>
<td>50</td>
<td>No</td>
<td>14</td>
<td>No</td>
</tr>
<tr>
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<td>Atrial pacing</td>
<td>44</td>
<td>−20</td>
<td>60</td>
<td>No</td>
<td>30</td>
<td>No</td>
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<td>5</td>
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<td>−6</td>
<td>35</td>
<td>No</td>
<td>26</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>Conversion from AVNRT</td>
<td>55</td>
<td>−2</td>
<td>65</td>
<td>No</td>
<td>22</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>Atrial pacing</td>
<td>46</td>
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<td>54</td>
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</tr>
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<td>−26</td>
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<td>No</td>
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<td>9</td>
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<td>No</td>
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<tr>
<td>10</td>
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<td>−16</td>
<td>34</td>
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<td>104</td>
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<tr>
<td>11</td>
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<td>−20</td>
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<td>No</td>
<td>94</td>
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<tr>
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<td>46</td>
<td>−30</td>
<td>62</td>
<td>No</td>
<td>6</td>
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</table>

Mean±SD 43±7 −15±8 48±18 42±31

AVNRT indicates atrioventricular nodal reentrant tachycardia; H-V, His-ventricular; PP-QRS, interval from Purkinje potential to QRS; and VT, ventricular tachycardia.

Limitations
Intracardiac echocardiography is limited in terms of its spatial accuracy and resolution. Although intracardiac echocardiography was not always able to clearly visualize the ablation catheter located on the PMs during radiofrequency application, all

![Figure 4. A, Ablation sites on the anterior papillary muscles (APM). B, Change in QRS morphology in patients with APM-fascicular ventricular tachycardia (FVT). Subtle changes, especially in the limb leads, were observed during ablation (patient number 9).](image-url)
Figure 5. Local ventricular electrograms and intracardiac echo images at the successful ablation site of papillary muscle (PM)-fascicular ventricular tachycardia (FVT). A, Ventricular tachycardia (VT) during the initial procedure exhibited right bundle branch block (RBBB) configuration and superior right axis deviation. The left posterior fascicular region was ablated in the initial procedure. The patient had recurrent VT exhibiting RBBB configuration and horizontal axis. B, The local electrogram at the successful ablation site during the repeat procedure is shown. Both diastolic and presystolic Purkinje potentials (P1 and P2) were sequentially recorded during VT. C, The successful ablation site was located on the posterior papillary muscles (PPM), which was confirmed by real-time intracardiac ultrasound image (patient number 1).
ablation sites were confirmed in the 3D mapping system with respect to the PMs’ geometry that was reconstructed before radiofrequency application. However, the precise identification of small anatomic structures, such as fibromuscular bands and small PMs, is difficult using intracardiac echocardiography.

This was a retrospective study with a relatively small number of patients because of the rarity of PM-FVT. Further studies enrolling a higher number of patients with PM-FVT are needed to investigate its exact mechanism and to evaluate the safety and efficacy of catheter ablation for this arrhythmia.

Nevertheless, this study provides important findings that have therapeutic value. We now recognize that verapamil-sensitive FVT may originate from the Purkinje network lying around the PMs. It is important to consider mapping not only the left posterior and anterior fascicular regions but also around the PMs.

Conclusions
PM-FVT is a distinct entity, which involves the Purkinje network around the PMs in its reentrant circuit, and is characterized by distinctive electrocardiographic characteristics and
less sensitivity to verapamil administration compared with the common type of FVT. Ablation targeting the mid-diastolic Purkinje potentials that are recorded around the PMs during tachycardia can be effective in suppressing this arrhythmia.

Acknowledgments

We thank Osamu Igawa for providing us with macroscopic pictures illustrating the myocardial structure between the anterior and posterior papillary muscles.

Disclosures

Dr Nogami received honoraria from Medtronic and St. Jude Medical, illustrating the myocardial structure between the anterior and posterior papillary muscles.

References

Fascicular Ventricular Tachycardia Originating From Papillary Muscles: Purkinje Network Involvement in the Reentrant Circuit
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